


CLINICAL INVESTIGATIONS

Pericarditis among giant cell arteritis patients: From myth to reality

Shmuel Tiosano^{1,2}  | Yehuda Adler^{2,3} | Shir Azrielant^{1,2} | Yarden Yavne^{1,2} | Omer Gendelman^{1,2} | Dana Ben-Ami Shor^{1,2} | Doron Comaneshter⁴ | Guy Shalom^{5,6} | Arnon D. Cohen^{4,7} | Howard Amital^{1,2}

¹Department of Medicine B and Zabudowicz Center for Autoimmune Diseases, Sheba Medical Center, Tel-Hashomer, Ramat-Gan, Israel

²Sackler Faculty of Medicine, Tel-Aviv University, Tel-Aviv, Israel

³Management, Sheba Medical Center, Tel-Hashomer, Ramat-Gan, Israel

⁴Chief Physician's Office, Clalit Health Services, Tel-Aviv, Israel

⁵Department of Dermatology and Venereology, Soroka Medical Center, Beer-Sheva, Israel

⁶Faculty of Health Sciences, Ben-Gurion University of the Negev, Beer-Sheva, Israel

⁷Sial Research Center for Family Medicine and Primary Care, Faculty of Health Sciences, Ben Gurion University of the Negev, Beer Sheva, Israel

Correspondence

Howard Amital, MD, MHA, Department of Medicine B, Sheba Medical Center, Tel-Hashomer, 5262100, Israel
Email: howard.amital@sheba.health.gov.il

Background: Giant cell arteritis (GCA) is an inflammatory disease of unknown etiology affecting adults age > 50 years. GCA (also known as temporal arteritis) is a vasculitis of large and medium-size vessels that involves the extracranial branches of the carotid artery. Common manifestations include constitutional symptoms, headache, jaw claudication, scalp tenderness, and vision loss. Cardiac involvement in GCA is considered to be as low as 5%, and < 30 cases of pericarditis among GCA patients have been reported in the literature. The aim of this study was to evaluate the association between GCA and pericarditis by conducting a cross-sectional study utilizing the database of the largest healthcare provider in Israel.

Hypothesis: GCA is associated with pericarditis.

Methods: The proportion of past documentation of pericarditis among patients diagnosed with GCA was compared with that of their age- and sex-matched controls. Univariate analysis was performed using the χ^2 and t tests; multivariate analysis was performed using logistic regression.

Results: The study included 4329 GCA patients and 21 611 controls. GCA patients had higher rates of cardiovascular risk factors. Pericarditis was observed in 53 GCA patients and 72 controls (1.22% vs 0.33%, respectively; $P < 0.001$), significantly higher among GCA patients in comparison with controls. A significant interaction was found between GCA, pericarditis, and young age (<70 years).

Conclusions: The study showed an independent association between GCA and pericarditis, especially among young patients. Proper screening should be applied whenever a suspicion arises as to the existence of comorbidity in patients with either disease.

KEYWORDS

Giant Cell Arteritis, Inflammation, Pericarditis, Temporal Arteritis, Vasculitis

1 | INTRODUCTION

Giant cell arteritis (GCA), also known as temporal arteritis, is a vasculitis of large and medium-size vessels that commonly involves the extracranial branches of the carotid artery. The onset of GCA tends to be subacute; its manifestations are related to the ischemia that might result. GCA has been associated with constitutional symptoms (fever, malaise, fatigue), as well as headache, jaw claudication, scalp tenderness, and loss of vision.¹ The incidence peaks between the ages of 70 to 79 and appearance before the age of 50 is rare.²

An article written in 1980 by How et al³ argued that although GCA has been associated with various cardiological conditions, such as myocardial infarction and congestive heart failure, the disease is often overlooked by cardiologists. One of the cardiological manifestations that was mentioned in their study was pericarditis.

Pericarditis, in common with GCA, is also an inflammatory process, but one that mainly involves the pericardial sac. Its etiology and underlying mechanism are mostly unknown; infections and autoimmune and autoinflammatory processes have all been suggested playing a role in the development of the idiopathic form of the disease.^{4,5}

It is characterized by a sharp and severe pleuritic chest pain and can also present with constitutional symptoms. Other manifestations include specific electrocardiographic findings and a pericardial friction rub heard on physical examination, which is pathognomonic for the disease.

Although evidence supporting the association between GCA and pericarditis has been accumulating for more than half a century, it is still often overlooked today.⁶ Only several case studies have reported pericardial effusions in patients diagnosed with GCA,^{7–11} and many of these patients were presented due to unusual manifestations of 1 of the 2 diseases. Overall, there are about 2 dozen case reports to date,¹² and the interest in this association seems to have subsided in recent years.

In this study, we wished to rekindle the investigation into the association between these 2 conditions and to examine it in a large, population-based database. The grand scale of this study will help shed light on the true nature of this association.

2 | METHODS

This study is one of a series of explorative and analytic studies based on the chronic disease registry of Clalit Health Services (CHS), the largest healthcare provider in Israel. We performed a “big-data” analysis by utilizing the medical database of CHS. CHS has a comprehensive computerized database and a chronic disease registry, both of which receive real-time input from pharmaceutical, medical, and administrative computerized operating systems in a continuous manner. The CHS chronic disease registry is composed of diagnoses of different medical conditions derived from records of community physicians or from hospital discharge letters.

The study was designed as a cross-sectional study, as depicted in Figure 1. The proportion of documented history of pericarditis was compared between patients with GCA and their age- and sex-matched controls. GCA patients were defined as patients who had documented diagnosis of GCA at least once in their medical records as outpatients by a community physician, either a primary-care physician or a specialist, or who were diagnosed with GCA in their hospital discharge papers as inpatients (or both as inpatients and outpatients, when appropriate). Pericarditis was defined similarly, but specifically

using the *International Classification of Diseases* codes 393, 420, and 423, and their subdivisions. All GCA patients detected in the CHS database were included in this study. Controls were randomly selected from the CHS database, with the exclusion of GCA patients. Five controls were matched by age and sex for each GCA patient (Figure 1). Age matching was based on year of birth. We decided to limit the age of subjects in the study to 85 (born in 1933 or later) due to a significant age disparity between GCA patients and controls. This inequality was caused by the fact that many controls who developed comorbidities were deceased; hence, their age was lower at the time of study conduction as compared with their GCA counterparts. The problematic issue of recruiting elderly controls was described before by Salthouse.¹³

Data available from the CHS database included age, sex, body mass index (BMI), socioeconomic status (SES), as well as diagnoses of chronic diseases, such as hypertension, hyperlipidemia, and smoking status (ever smoked). Due to the limitations of the database, it was not possible to determine the etiology of each disorder. However, the validity of the diagnoses in the registry was found to be high in previous studies.^{14–16}

2.1 | Statistical analysis

The proportion of pericarditis and other covariates was compared between GCA patients and controls in the study sample group, and it was also analyzed independently to seek for interactions across all strata of categorical and continuous variables. For the matter of interactions, age variable was treated as dichotomous (age < 70 and ≥ 70). The cutoff was based on a review by Gonzalez-Gay et al. that stated that incidence of GCA is rising after age 70.² The χ^2 test was used to assess the distribution of categorical variables between GCA patients and controls, and the t test was applied for continuous variables. The association between pericarditis and GCA was evaluated by a multivariate logistic regression model. Odds ratios (ORs), as well as 95% confidence intervals (CIs), are presented. Statistical analysis was performed using R Statistical Software version 3.3.0 (R Foundation for Statistical Computing, Vienna, Austria).

The study was approved by the ethics committee of CHS, located in Soroka Medical Center, Be'er Sheva, Israel.

3 | RESULTS

The study included 4329 GCA patients and 21 611 controls (Table 1). The mean ages of the GCA patients and controls were 65.9 and 64.1 years, respectively. Both groups had a clear female predominance and were overweight according to their mean BMI values. GCA patients presented significantly more cardiovascular risk factors, including smoking. Pericarditis was observed in 53 GCA patients vs 72 controls, producing a significantly higher rate of pericarditis among GCA patients in comparison with controls (1.22% vs 0.33%, respectively; $P < 0.001$). GCA patients also exhibited a greater proportion of high SES when compared with controls.

A significant interaction between GCA and pericarditis was observed among all strata of study covariates, namely younger age

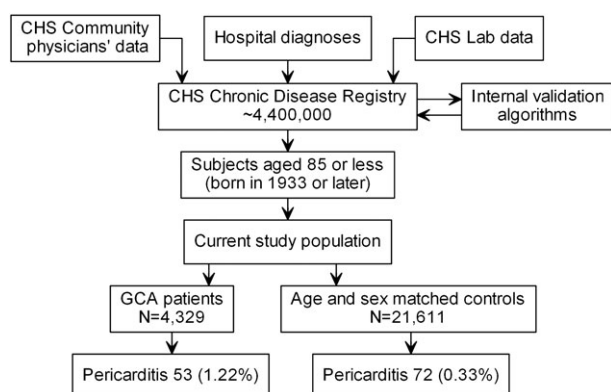


FIGURE 1 Study flowchart. Abbreviations: CHS, Clalit Health Services; GCA, giant cell arteritis

TABLE 1 Basic characteristics of GCA patients and matched controls

Characteristic	Controls (No GCA), N = 21 611	GCA, N = 4329	P Value
Age, y	64.1 ± 14.2	65.9 ± 14.1	<0.001
Female sex	14 817 (68.6)	2967 (68.5)	0.98
BMI, kg/m ²	28.4 ± 5.91	28.4 ± 5.70	0.08
SES			
Low	4051 (37.8)	1616 (37.4)	—
Medium	4361 (40.7)	1743 (40.4)	0.31
High	2302 (21.5)	958 (22.2)	<0.001
HTN	5648 (26.1)	2432 (56.2)	<0.001
Hyperlipidemia	7382 (34.2)	3223 (74.5)	<0.001
DM	3290 (15.2)	1396 (32.2)	<0.001
Smoking	3496 (16.2)	1578 (36.5)	<0.001
Pericarditis	72 (0.33)	53 (1.22)	<0.001

Abbreviations: BMI, body mass index; DM, diabetes mellitus; GCA, giant cell arteritis; HTN, hypertension; SD, standard deviation; SES, socioeconomic status. Data are presented as n (%) or mean ± SD.

(<70 years), except the following groups: low SES, BMI <25 kg/m², diabetes, and with and without smoking (Figure 2). In a multivariate logistic regression model (Table 2), GCA was found to be independently associated with pericarditis (OR: 1.69, 95% CI: 1.16–2.144). Smoking was also found to be independently associated with pericarditis. However, other covariates, such as age and sex, failed to demonstrate a similar independent association.

4 | DISCUSSION

In the current study, we explored the association between GCA and pericarditis; we demonstrated that even after adjustment for age, sex,

TABLE 2 Multivariate logistic regression: covariates associated with pericarditis

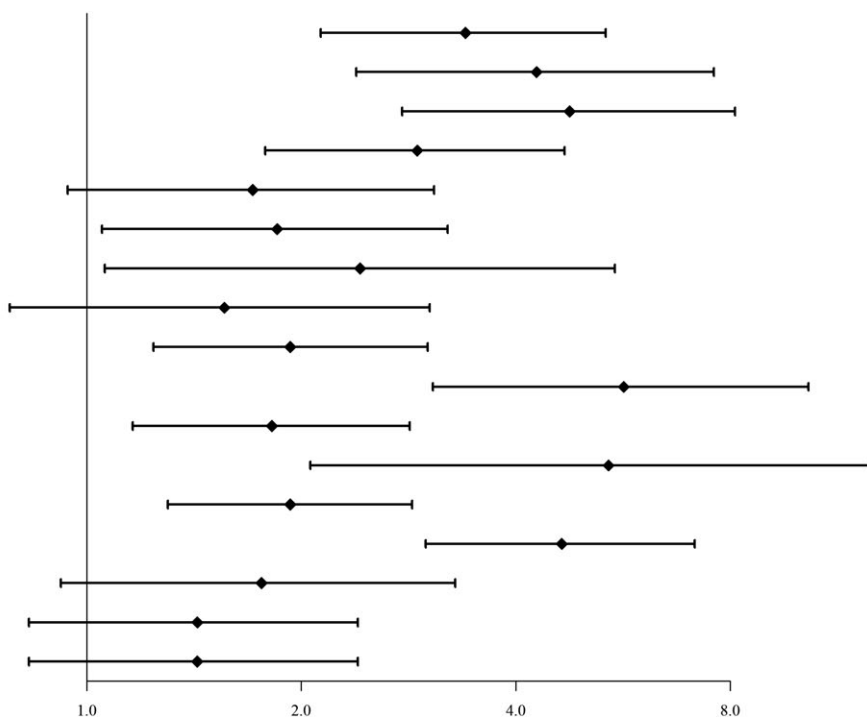
Characteristic	OR	95% CI	P Value
Age	1.01	1.00–1.03	0.15
Female sex	0.78	0.52–1.17	0.23
BMI, 1-kg/m ² increment	0.98	0.94–1.01	0.22
SES			
Medium vs low	0.89	0.59–1.35	0.58
High vs low	0.80	0.47–1.31	0.38
HTN	1.36	0.88–2.13	0.18
Hyperlipidemia	1.41	0.85–2.46	0.20
DM	0.85	0.56–1.29	0.46
Smoking	1.55	1.05–2.27	0.03
GCA	1.69	1.16–2.44	0.01

Abbreviations: BMI, body mass index; CI, confidence interval; DM, diabetes mellitus; GCA, giant cell arteritis; HTN, hypertension; OR, odds ratio; SD, standard deviation; SES, socioeconomic status.

and other cardiovascular risk factors was performed, the 2 diseases remained significantly associated. The association was especially prominent among younger (age < 70 years) GCA patients. For years, the existence of the association between GCA and pericarditis was supported only by anecdotal case reports. This population-based study is the first to quantify the proportion of pericarditis among GCA patients, as well as the first to establish an independent connection between the 2 diseases.

Overall, cardiac disease (endocardial, myocardial, or pericardial) is estimated to occur in about 5% of GCA patients.¹⁷ However, regarding pericardial involvement, the rates are uncertain. Our study has demonstrated that the rate of pericarditis among GCA patients was 1.22%, as opposed to a French study by Zenone and Puget,¹⁸ which

Gender: Female	3.40 [2.13;5.35]
Gender: Male	4.28 [2.39;7.59]
Age: 18–69	4.76 [2.77;8.12]
Age: 70+	2.91 [1.78;4.68]
SES: Low	1.71 [0.94;3.07]
SES: Medium	1.85 [1.05;3.21]
SES: High	2.42 [1.06;5.51]
BMI ≤ 25	1.56 [0.78;3.03]
BMI > 25	1.93 [1.24;3.01]
No Hypertension	5.67 [3.06;10.3]
Hypertension	1.82 [1.16;2.84]
No Hyperlipidemia	5.40 [2.06;12.6]
Hyperlipidemia	1.93 [1.30;2.86]
No Diabetes	4.64 [2.99;7.13]
Diabetes	1.76 [0.92;3.29]
No Smoking	1.43 [0.83;2.40]
Smoking	1.43 [0.83;2.40]

**FIGURE 2** Interaction between GCA and pericarditis by strata of study covariates. Abbreviations: BMI, body mass index; GCA, giant cell arteritis; SES, socioeconomic status

described pericardial effusion in 3.5% of patients. This discrepancy could be explained by the nature of the 2 disorders, which are uncommon and therefore are challenging to diagnose, even for experienced clinicians. The presentation of GCA may precede pericarditis or coincide with it; indeed, in some cases, pericarditis and even pericardial tamponade were the first overt manifestations of GCA.¹⁹

Pericarditis is a term used to describe a group of diseases arising from different etiologies. Autoimmune pericarditis, which may occur in rheumatoid arthritis or systemic lupus erythematosus, is mediated by an antibody-provoked interferon response.²⁰ On the other hand, an autoinflammatory systemic process such as GCA may induce pericarditis via the inflammasome pathway, in which the activation of sensor molecules triggers a predominant T helper 17 cell response that results in excessive interleukin-1 (IL-1) production.

Currently, high-dose corticosteroid therapy is still considered the mainstay of pharmacotherapy in GCA, which holds a great number of side effects. However, in recent years, evidence is accumulating toward the role of IL-1 inhibitors, such as anakinra and canakinumab, in the treatment of GCA. Presently, IL-1 inhibitors are labeled for rheumatoid arthritis, juvenile idiopathic arthritis, and autoinflammatory conditions such as familial Mediterranean fever. Several small studies that have attempted to treat refractory GCA with IL-1 inhibitors have enjoyed relative success.^{21,22} Although Imazio et al²³ demonstrated the efficacy of using colchicine and anti-inflammatory drugs for the treatment of acute pericarditis, IL-1 inhibitors were also tried as a novel agent for treating idiopathic recurrent pericarditis with a high rate of remission, but only in small-scale studies as well.²⁴ It is only reasonable to believe that in the future, IL-1 inhibitors will become key players in the treatment of pericarditis.²⁵ Therefore, IL-1 seems to be involved in both diseases, and might, at least partly, explain their common pathways. Interestingly, the IL-6 receptor α inhibitor tocilizumab showed promising results in treating GCA, with longer to glucocorticoid-free remission compared with placebo.²⁶ This might open a door for future IL-6 pathway inhibition in also treating pericarditis.

A review by Cantarini et al²⁷ states that GCA is one of the systemic diseases that may cause idiopathic recurrent pericarditis on top of acute pericarditis. The partially common pathogenic pathways and shared response to pharmacotherapy of GCA and pericarditis may explain the association between the 2 disorders that was demonstrated in our study. Another plausible explanation for the increased rate of pericarditis among GCA patients is their compromised immune state due to immunosuppressive therapy, leading to higher susceptibility to opportunistic infections. The resulting postviral pericarditis may require treatment with nonsteroidal anti-inflammatory drugs or colchicine.²⁰

Our study showed a significant interaction between GCA, pericarditis, and younger age (age < 70 years). These findings correspond well with most case reports and case series previously published on the subject.^{8,9,19,28–31} However, several articles have reported cases of adults age > 70 years with simultaneous presentations of both GCA and pericarditis, which usually regressed after corticosteroid treatment.^{12,32–36} A Spanish case report described a 76-year-old female patient with pectoral girdle pain and a pleural effusion on x-ray who was eventually diagnosed with polymyalgia rheumatica (PMR)

accompanied by pleuropericarditis and whose high levels of CA-125 indicated serosal involvement, rather than malignancy.³⁷ Based on these observations, it has been suggested that while investigating idiopathic pericarditis among elderly patients, a temporal artery biopsy should be obtained even in the absence of classical GCA manifestations.³⁸ Current guidelines³⁹ note that pericarditis, acute or recurrent, reflects the presence of systemic autoimmune disease in 5% to 15% of the cases; in such suspected cases, the authors warrant a targeted search for etiology and recommend gaining rapid control over the primary disease.

The strength of our study is derived from its large scale, and its novelty; it is the first study to date to describe comorbidity of GCA and pericarditis in such a large number of patients. Our study also provides a good characterization of the patients who suffer from both diseases, as the diagnoses were done in the setting of both community and hospitals, and the computerized data registration started approximately at the beginning of the millennium until the year 2016, when data for the current study were collected.

The clinical significance of this study lies in its ability to shed light on GCA as an often-forgotten etiology for pericarditis, especially among adults age < 70 years. GCA may be a challenging diagnosis; physicians should consider it in some difficult cases of pericardial diseases, including probably unexplained chronic effusions with elevated CRP; in these cases, a computed tomography scan may demonstrate the thickening of large-vessel walls and positron emission tomography may show some capitation at the level of vessel walls.

4.1 | Study limitations

Our study does have several drawbacks. The main limitation is that the diagnoses of both GCA and pericarditis were not based on standardized diagnostic criteria, but rather on diagnostic labels drawn from CHS physician reports. However, previous studies have supported the high validity of the diagnoses in this database.⁴⁰ In this study, past diagnosis of pericarditis represented either acute pericarditis or chronic pericarditis, or pleural effusion (or a combination), which may arise from different etiologies. Another possible disadvantage of this study is that it was designed as a cross-sectional study, so it was impossible to determine whether pericarditis occurred before, during, or after the diagnosis of GCA. Hence, this theoretically prevents the discussion of causal relationship between GCA and pericarditis; however, as discussed previously, pericarditis may be the first presentation of an occult GCA. Although PMR is closely related to GCA, it was not included in this study due to the low documentation rate of this comorbidity alongside GCA in the CHS database. In a previous study we discussed this discrepancy, which is due to registration and documentation habits in real life, rather than lower-than-expected rates of PMR.⁴⁰

5 | CONCLUSION

The current study has shown that GCA is independently associated with pericarditis, especially among patients age < 70 years. Proper

screening methods should be applied whenever suspicion arises to the existence of comorbidity in patients with either disease.

Author contributions

Arnon D. Cohen, MD, and Howard Amital, MD, share equal contribution.

Conflicts of interest

The authors declare no potential conflicts of interest.

ORCID

Shmuel Tiosano  <http://orcid.org/0000-0002-1748-4297>

REFERENCES

- Hunder GG, Bloch DA, Michel BA, et al. The American College of Rheumatology 1990 criteria for the classification of giant cell arteritis. *Arthritis Rheum*. 1990;33:1122–1128.
- Gonzalez-Gay MA, Vazquez-Rodriguez TR, Lopez-Diaz MJ, et al. Epidemiology of giant cell arteritis and polymyalgia rheumatica. *Arthritis Rheum*. 2009;61:1454–1461.
- How J, Strachan RW, Bewsher PD. Giant cell arteritis—a cardiological blind spot? *Am Heart J*. 1980;100:405–407.
- Imazio M, Adler Y. Management of pericardial effusion. *Eur Heart J*. 2013;34:1186–1197.
- Imazio M, Gaita F, LeWinter M. Evaluation and treatment of pericarditis: a systematic review [published corrections appear in JAMA. 2015; 314:1978 and JAMA. 2016;315:90]. *JAMA*. 2015;314:1498–1506.
- Knockaert DC. Cardiac involvement in systemic inflammatory diseases. *Eur Heart J*. 2007;28:1797–1804.
- Stanley D, Henderson D, Harris S. Giant cell arteritis associated with pericarditis and large vessel disease. *Aust N Z J Med*. 1991;21: 353–355.
- Clementz GL, Gold F, Khaizer N, et al. Giant cell arteritis associated with pericarditis and pancreatic insufficiency in a patient with psoriatic arthritis. *J Rheumatol*. 1989;16:128–129.
- Dupond JL, Leconte des Floris R. Temporal arteritis manifested as an acute febrile pericarditis. *JAMA*. 1982;247:2371–2372.
- Malmvall BE. Chapter 6: involvement of different organs in giant cell arteritis. *Acta Med Scand*. 1982;211:29–32.
- Imazio M. Pericardial involvement in systemic inflammatory diseases. *Heart*. 2011;97:1882–1892.
- Bablekos GD, Michaelides SA, Karachalios GN, et al. Pericardial involvement as an atypical manifestation of giant cell arteritis: report of a clinical case and literature review. *Am J Med Sci*. 2006;332: 198–204.
- Salthouse TA. All data collection and analysis methods have limitations: reply to Rabbitt (2011) and Lindenberger (2011). *Psychol Bull*. 2011;137:796–799.
- Tiosano S, Nir Z, Gendelman O, et al. The association between systemic lupus erythematosus and bipolar disorder—a big data analysis. *Eur Psychiatry*. 2017;43:116–119.
- Versini M, Tiosano S, Comaneshter D, et al. Smoking and obesity in systemic lupus erythematosus: a cross-sectional study. *Eur J Clin Invest*. 2017;47:422–427.
- Merdler-Rabinowicz R, Tiosano S, Comaneshter D, et al. Comorbidity of gout and rheumatoid arthritis in a large population database. *Clin Rheumatol*. 2017;36:657–660.
- Miloslavsky E, Unizony S. The heart in vasculitis. *Rheum Dis Clin North Am*. 2014;40:11–26.
- Zenone T, Puget M. Pericardial effusion and giant cell arteritis. *Rheumatol Int*. 2014;34:1465–1469.
- Pant I, Smith A, Thomas T, et al. A woman with progressive lethargy and sudden onset of shortness of breath. *Clin Med (Lond)*. 2013;13: 625–627.
- Xu B, Harb SC, Cremer PC. New insights into pericarditis: mechanisms of injury and therapeutic targets. *Curr Cardiol Rep*. 2017;19:60.
- Ly KH, Stirnemann J, Liozon E, et al. Interleukin-1 blockade in refractory giant cell arteritis. *Joint Bone Spine*. 2014;81:76–78.
- Cavalli G, Dinarello CA. Treating rheumatological diseases and co-morbidities with interleukin-1 blocking therapies. *Rheumatology (Oxford)*. 2015;54:2134–2144.
- Imazio M, Brucato A, Cemin R, et al. A randomized trial of colchicine for acute pericarditis. *N Engl J Med*. 2013;369:1522–1528.
- Brucato A, Imazio M, Gattorno M, et al. Effect of anakinra on recurrent pericarditis among patients with colchicine resistance and corticosteroid dependence: the AIRTRIP randomized clinical trial. *JAMA*. 2016;316:1906–1912.
- Azrielant S, Shoenfeld Y, Adler Y. Recurrent pericarditis: is immunotherapy the answer? *Isr Med Assoc J*. 2018;20:190–191.
- Stone JH, Tuckwell K, Dimonaco S, et al. Trial of tocilizumab in giant-cell arteritis. *N Engl J Med*. 2017;377:317–328.
- Cantarini L, Imazio M, Brizi MG, et al. Role of autoimmunity and auto-inflammation in the pathogenesis of idiopathic recurrent pericarditis. *Clin Rev Allergy Immunol*. 2013;44:6–13.
- Guillaume M, Vachier F, Cogan E. Pericarditis: an unusual manifestation of giant cell arteritis. *Am J Med*. 1991;91:662–664.
- Nicolás Sánchez FJ, Torres Cortada G, Sarrat Nuevo RM, et al. Pericarditis and giant cell arteritis [article in Spanish]. *An Med Interna Madr Spain* 1984. 2006;23:382–384.
- Couturier B, Huyge V, Soyfoo MS. Pericarditis revealing large vessel vasculitis. *ISRN Rheumatol*. 2011;2011:648703.
- Daumas A, Rossi P, Jacquier A, Granel B. Myopericarditis revealing giant cell arteritis in the elderly. *J Rheumatol*. 2012;39:665–666.
- Guindon A, Rossi P, Bagneres D, et al. Pericarditis: a giant cell arteritis manifestation [article in French]. *Rev Med Interne*. 2007;28:326–331.
- Moulis G, Sailer L, Astudillo L, et al. Péricardite inaugurale d'une maladie de Horton [article in French]. *Rev Med Interne*. 2010;31:46–48.
- Blot M, Guépet H, Aubriot-Lorton MH, et al. An atypical case of giant cell arteritis (Horton's disease) associated with facial swelling, confusion, and pericarditis in an elderly woman. *J Am Geriatr Soc*. 2010;58: 2040–2041.
- Matsue Y, Ohno M, Nagahori W, et al. A case of giant cell arteritis with massive pericardial effusion. *Heart Vessels*. 2011;26:562–564.
- Tasliyurt T, Sivgin H, Bekar L, et al. A rare cause of pericardial effusion: giant cell arteritis. *Case Rep Rheumatol*. 2014;2014:1–3.
- Jarana Aparicio I, Pedraza Serrano F, de Miguel Díez J. Pleural and pericardial effusion in a patient with polymyalgia rheumatica [article in English, Spanish]. *Arch Bronconeumol*. 2014;50:370–371.
- Daumas A, Bernard F, Rossi P, et al. “Extra-Cranial” Manifestations of Giant Cell Arteritis, Advances in the Diagnosis and Treatment of Vasculitis. Dr. Luis M Amezcua-Guerra (Ed.), ISBN: 978-953-307-786-4, InTech, 2011. Available from: <http://www.intechopen.com/books/advances-in-the-diagnosis-and-treatment-of-vasculitis/-extra-cranial-manifestations-of-giant-cell-arteritis>
- Adler Y, Charron P, Imazio M, et al. 2015 ESC Guidelines for the diagnosis and management of pericardial diseases: the Task Force for the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC). Endorsed by the European Association for Cardio-Thoracic Surgery (EACTS). *Eur Heart J*. 2015;36: 2921–2964.
- Yavne Y, Tiosano S, Watad A, et al. Association between giant cell arteritis and thyroid dysfunction in a “real life” population. *Endocrine*. 2017;57:241–246.

How to cite this article: Tiosano S, Adler Y, Azrielant S, et al. Pericarditis among giant cell arteritis patients: From myth to reality. *Clin Cardiol*. 2018;41:623–627. <https://doi.org/10.1002/clc.22927>